IN THE CLAIMS:

Please cancel claims 116, 117 and 120, without prejudice. Please amend the claims and add new claims 121 to 155 as indicated on the following listing of claims:

1.-30. (Cancelled)

31. (Currently Amended) A method of treating a mammalian subject having diabetes comprising contacting ~~transforming gut mucosal tissue endocrine cells, or~~ gastrointestinal mucosal tissue ~~endocrine~~ cells~~, or gut mucosal tissue stem cells, pluripotent or multipotent progenitor cells, or gastrointestinal mucosal~~ comprising K cells or stem cells, ~~pluripotent~~ or multipotent progenitor cells that differentiate into K cells in the subject with a polynucleotide vector comprising a glucose-dependent insulinotropic polypeptide (GIP) promoter ~~or chromogranin A promoter~~ in operable linkage with a nucleic acid encoding insulin, wherein said ~~transforming~~ contacting occurs *in vivo* via intra-cavity delivery to stomach or small intestine, thereby producing transformed ~~mucosal~~ K cells or stem cells, or multipotent progenitor cells that differentiate into K cells, and wherein orally feeding ~~contacting said transformed mucosal tissue cells in~~ the subject ~~with~~ an amount of ~~sugar,~~ glucose, sucrose, fructose, carbohydrate, polypeptide, amino acid or fat ~~induces~~ increases transcription or secretion of the insulin by the transformed ~~mucosal tissue~~ cells in an amount effective to decrease blood glucose in the subject, thereby treating the mammalian subject having diabetes.

32.-33. (Cancelled)

34. (Previously Presented) The method of claim 31, wherein the diabetes comprises type 1 diabetes.

35. (Currently Amended) The method of claim 31, wherein the subject has a fasting plasma glucose level greater than 110 mg/dl prior to treatment.

36. (Previously Presented) The method of claim 31, wherein the diabetes comprises insulin-independent (type 2) diabetes.

37. (Cancelled)

38. (Currently Amended) The method of claim 31, wherein the ~~sugar~~ glucose increases transcription and secretion of the insulin by the transformed cells.

39. (Cancelled)

40. (Currently Amended) The method of claim 31, wherein the polypeptide or amino acid increases secretion ~~transcription~~ of the insulin ~~is increased in~~ by the transformed cells.

41.-42. (Cancelled)

43. (Currently Amended) The method of claim 31, wherein the glucose-dependent insulinotropic polypeptide (GIP) promoter ~~or chromogranin A promoter~~ comprises a functional variant or a functional subsequence thereof, and wherein the glucose-dependent insulinotropic polypeptide (GIP) promoter ~~or chromogranin A promoter~~ functional variant or subsequence retains all or a part of non-variant or full-length glucose-dependent insulinotropic polypeptide (GIP) promoter ~~or chromogranin A promoter expression~~ transcription function.

44.-46. (Cancelled)

47. (Currently Amended) The method of claim 31, wherein the ~~mucosal tissue endocrine cell or mucosal tissue~~ transformed K cells, stem cells, ~~pluripotent~~ or multipotent progenitor cells is present in the small intestine.

48. (Cancel)

49. (Currently Amended) The method of claim 31, wherein the ~~mucosal tissue endocrine cell or mucosal tissue~~ transformed K cells, stem cells, ~~pluripotent~~ or multipotent progenitor cells is present in the stomach.

50. (Cancelled)

51. (Currently Amended) The method of claim 31, wherein ~~the mucosal tissue endocrine~~ said contacting produces transformed K cells ~~cell is a K-cell, L-cell, S-cell, G-cell, D-cell, I-cell, Mo-cell, GR cell or entero-endocrine cell~~.

52. (Cancelled)

53. (Cancelled)

54. (Currently Amended) The method of claim 31, wherein the glucose-dependent insulinotropic polypeptide (GIP) promoter ~~or chromogranin A promoter~~ in operable linkage with [[a]] the nucleic acid encoding insulin further comprises a vector.

55. (Previously Presented) The method of claim 54, wherein the vector comprises a viral vector.

56.-70. (Cancelled)

71. (Currently Amended) A method of ~~treating~~ reducing blood glucose in a mammalian subject having undesirable body mass or obesity comprising contacting ~~transforming gut mucosal tissue endocrine cells, or~~ gastrointestinal mucosal tissue ~~endocrine~~ cells~~, or gut mucosal tissue stem cells, pluripotent or multipotent progenitor cells, or gastrointestinal mucosal~~ comprising K cells or stem cells, ~~pluripotent~~ or multipotent progenitor cells that differentiate into K cells in the subject with a polynucleotide vector comprising a glucose-dependent insulinotropic polypeptide (GIP) promoter ~~or chromogranin A promoter~~ in operable linkage with a nucleic acid encoding leptin, wherein said contacting ~~transforming~~ occurs *in vivo* via intra-cavity delivery to stomach or small intestine, thereby producing transformed ~~mucosal tissue~~ K cells or stem cells, or multipotent progenitor cells that differentiate into K cells, and wherein orally feeding ~~contacting said transformed mucosal tissue cells in~~ the subject ~~with~~ an amount of ~~sugar,~~ glucose, sucrose, fructose, carbohydrate, polypeptide, amino acid or fat ~~induces~~ increases transcription or secretion of the leptin by the transformed cells in an amount effective to ~~treat undesirable body mass or obesity~~ reduce blood glucose in the subject.

72. (Currently Amended) The method of claim 71, wherein the ~~undesirable body mass or obesity is reduced~~ subject is obese.

73. (Currently Amended) The method of claim 71, wherein the ~~sugar~~ glucose increases transcription and secretion of the leptin by the transformed cells.

74.-75. (Cancelled)

76. (Currently Amended) The method of claim 71, wherein the glucose-dependent insulinotropic polypeptide (GIP) promoter ~~or chromogranin A promoter~~ comprises a functional variant or functional subsequence thereof that retains all or a part of non-variant or full-length glucose-dependent insulinotropic polypeptide (GIP) promoter ~~or chromogranin A promoter expression~~ transcription function.

77. (Cancelled)

78. (Currently Amended) The method of claim 71, wherein the ~~mucosal tissue endocrine cell or mucosal tissue~~ transformed K cells, stem cells, ~~pluripotent~~ or multipotent progenitor cells is present in the small intestine.

79. (Cancelled)

80. (Currently Amended) The method of claim 71, wherein the ~~mucosal tissue endocrine cell or mucosal tissue~~ transformed K cells, stem cells, ~~pluripotent~~ or multipotent progenitor cells is present in the stomach.

81. (Cancelled)

82. (Currently Amended) The method of claim 71, wherein ~~the mucosal tissue endocrine cell~~ said contacting produces transformed K cells ~~is a K-cell, L-cell, S-cell, G-cell, D-cell, I-cell, Mo-cell, GR-cell or entero-endocrine cell~~.

83.-84. (Cancelled)

85. (Currently Amended) The method of claim 71, wherein the glucose-dependent insulinotropic polypeptide (GIP) promoter ~~or chromogranin A promoter~~ in operable linkage with [[a]] the nucleic acid encoding leptin further comprises a vector.

86. (Previously Presented) The method of claim 85, wherein the vector comprises a viral vector.

87. (Currently Amended) The method of claim 31, wherein said ~~transforming~~ contacting *in vivo* via intra-cavity delivery is with an endoscope, feeding tube, cannula, or catheter.

88. (Currently Amended) The method of claim 71, wherein said ~~transforming~~ contacting *in vivo* via intra-cavity delivery is with an endoscope, feeding tube, cannula, or catheter.

89.-113. (Cancelled)

114. (Currently Amended) The method of claim 31, wherein said ~~transforming~~ contacting *in vivo* via intra-cavity delivery occurs orally.

115. (Currently Amended) The method of claim 71, wherein said ~~transforming~~ contacting *in vivo* via intra-cavity delivery occurs orally.

116.-117. (Cancelled)

118. (Currently Amended) The method of claim 54, wherein said vector comprises a vector that facilitates integration of the nucleic acid encoding insulin into the genome of said transformed ~~mucosal tissue endocrine cells~~ K cells, stem cells, or multipotent progenitor cells.

119. (Currently Amended) The method of claim 71, wherein said vector comprises a vector that facilitates integration of the nucleic acid encoding leptin into the genome of said transformed ~~mucosal tissue endocrine cells~~ K cells, stem cells, or multipotent progenitor cells.

120. (Cancelled)

121. (New) A method of treating a mammalian subject having diabetes comprising contacting gastrointestinal mucosal tissue cells comprising gut endocrine cells, or stem cells, or multipotent progenitor cells that differentiate into gut endocrine cells in the subject with a polynucleotide vector comprising a chromogranin A promoter in operable linkage with a nucleic acid encoding insulin, wherein said contacting occurs *in vivo* via intra-cavity delivery to stomach or small intestine, thereby producing transformed gut endocrine cells, or stem cells, or multipotent progenitor cells that differentiate into gut endocrine cells in stomach or small intestine, and wherein orally feeding the subject an amount of glucose, carbohydrate, polypeptide, amino acid or fat increases secretion of the insulin by transformed cells in an amount effective to decrease blood glucose in the subject.

122. (New) The method of claim 121, wherein the diabetes comprises type 1 diabetes.

123. (New) The method of claim 121, wherein the subject has a fasting plasma glucose level greater than 110 mg/dl prior to treatment.

124. (New) The method of claim 121, wherein the diabetes comprises insulin-independent (type 2) diabetes.

125. (New) The method of claim 121, wherein the chromogranin A promoter comprises a functional variant or a functional subsequence thereof, and wherein the chromogranin A promoter functional variant or subsequence retains all or a part of non-variant or full-length or chromogranin A promoter transcription function.

126. (New) The method of claim 121, wherein the transformed gut endocrine cells, or stem cells, or multipotent progenitor cells is present in the small intestine.

127. (New) The method of claim 121, wherein the transformed gut endocrine cells, or stem cells, or multipotent progenitor cells is present in the stomach.

128. (New) The method of claim 121, wherein the chromogranin A promoter in operable linkage with the nucleic acid encoding insulin further comprises a vector.

129. (New) The method of claim 128, wherein the vector comprises a viral vector.

130. (New) The method of claim 121, wherein said contacting *in vivo* via intra-cavity delivery is with an endoscope, feeding tube, cannula, or catheter.

131. (New) The method of claim 121, wherein said contacting *in vivo* via intra-cavity delivery occurs orally.

132. (New) The method of claim 121, wherein said vector comprises a vector that facilitates integration of the nucleic acid encoding insulin into the genome of said transformed gut endocrine cells, or stem cells, or multipotent progenitor cells.

133. (New) A method of reducing blood glucose in a mammalian subject having undesirable body mass or obesity comprising contacting gastrointestinal mucosal tissue cells comprising gut endocrine cells, or stem cells, or multipotent progenitor cells that differentiate into gut endocrine cells in the subject with a polynucleotide vector comprising a chromogranin A promoter in operable linkage with a nucleic acid encoding leptin, wherein said contacting occurs *in vivo* via intra-cavity delivery to stomach or small intestine, thereby producing transformed gut endocrine cells, or stem cells, or multipotent progenitor cells that differentiate into gut endocrine cells, and wherein orally feeding the subject an amount of glucose, carbohydrate, polypeptide, amino acid or fat increases secretion of leptin by transformed cells in an amount effective to reduce blood glucose in the subject.

134. (New) The method of claim 133, wherein the subject is obese.

135. (New) The method of claim 133, wherein the chromogranin A promoter comprises a functional variant or functional subsequence thereof that retains all or a part of non-variant or full-length chromogranin A promoter transcription function.

136. (New) The method of claim 133, wherein the transformed gut endocrine cells, or stem cells, or multipotent progenitor cells is present in the small intestine.

137. (New) The method of claim 133, wherein the transformed gut endocrine cells, or stem cells, or multipotent progenitor cells is present in the stomach.

138. (New) The method of claim 133, wherein the gut endocrine cell is a K-cell, L-cell, S-cell, G-cell, D-cell, I-cell, Mo-cell, GR cell or entero-endocrine cell.

139. (New) The method of claim 133, wherein the chromogranin A promoter in operable linkage with the nucleic acid encoding leptin further comprises a vector.

140. (New) The method of claim 139, wherein the vector comprises a viral vector.

141. (New) The method of claim 133, wherein said contacting *in vivo* via intra-cavity delivery is with an endoscope, feeding tube, cannula, or catheter.

142. (New) The method of claim 133, wherein said contacting *in vivo* via intra-cavity delivery occurs orally.

143. (New) The method of claim 133, wherein said vector comprises a vector that facilitates integration of the nucleic acid encoding insulin into the genome of said transformed gut endocrine cells, or stem cells, or multipotent progenitor cells.

144. (New) A method of treating a mammalian subject having diabetes comprising contacting gastrointestinal mucosal tissue cells comprising K cells, or stem cells, or multipotent progenitor cells that differentiate into K cells in the subject with a polynucleotide vector comprising a proglucagon promoter in operable linkage with a nucleic acid encoding insulin, wherein said contacting occurs *in vivo* via intra-cavity delivery to stomach or small intestine, thereby producing transformed K cells or stem cells, or multipotent progenitor cells that differentiate into K cells, and wherein orally feeding the subject an amount of glucose, sucrose, fructose, carbohydrate, polypeptide, amino acid or fat increases secretion of insulin by the transformed cells in an amount effective to decrease blood glucose in the subject, thereby treating the mammalian subject having diabetes.

145. (New) The method of claim 144, wherein the diabetes comprises type 1 diabetes.

146. (New) The method of claim 144, wherein the subject has a fasting plasma glucose level greater than 110 mg/dl prior to treatment.

147. (New) The method of claim 144, wherein the diabetes comprises insulin-independent (type 2) diabetes.

148. (New) The method of claim 144, wherein the transformed K cells, stem cells, or multipotent progenitor cells is present in the small intestine.

149. (New) The method of claim 144, wherein the transformed K cells, stem cells, or multipotent progenitor cells is present in the stomach.

150. (New) The method of claim 144, wherein said contacting produces transformed K cells.

151. (New) The method of claim 144, wherein the proglucagon promoter in operable linkage with the nucleic acid encoding insulin further comprises a vector.

152. (New) The method of claim 151, wherein the vector comprises a viral vector.

153. (New) The method of claim 144, wherein said contacting *in vivo* via intra-cavity delivery is with an endoscope, feeding tube, cannula, or catheter.

154. (New) The method of claim 144, wherein said contacting *in vivo* via intra-cavity delivery occurs orally.

155. (New) The method of claim 144, wherein said vector comprises a vector that facilitates integration of the nucleic acid encoding insulin into the genome of said transformed K cells, stem cells, or multipotent progenitor cells.